INTRODUCTION

The prevalence of aortic valve disease is increasing with the aging of the population, with a reported incidence of approximately 3–4% in the adult population [1,2]. Aortic valve disease is the most common valvular disease requiring valve replacement. Although transcatheter aortic valve implantation (TAVI) shows good outcome for selected patients, aortic valve replacement (AVR) remains an equivalent treatment for patients with aortic valve stenosis or aortic valve regurgitation [2].

Despite much progress in surgical procedures and prosthetic valve design, complications after AVR significantly affect patient mortality; the reported 30-day and 1-year mortality rates are 2.1% and 4.9%, respectively [3]. The incidence of various complications after AVR is affected by valve type (mechanical vs. bioprosthetic), valve position, and clinical risk factors. Complications of AVR include paravalvular aortic regurgitation, dehiscence, infective endocarditis, aortic dissection, and hemolysis (Table 1).

Routine screening with echocardiography after aortic valve surgery is essential to detect postoperative or postprocedural complications. Echocardiography has several strengths, including ready availability, bedside examination suitability, and low cost. It provides information not only on the motion of the prosthetic valve, but also on functional information using Doppler imaging parameters, such as pressure gradients and flow velocity through the valve. However, echocardiography is an operator-dependent modality, and lesions can be obscured by prosthetic valve artifacts. To obtain additive information, computed tomography (CT) examination has been widely used. Cardiac CT imaging is a rapid, noninvasive, and accurate technique that enables clinicians to obtain a quick diagnosis and assists in treatment planning. However, CT does not provide real-time imaging, and beta-blocker medication is often necessary to reduce the heart rate during the CT acquisition due to the limited tem-
Echocardiography is a first-line tool for non-invasive study of cardiac diseases due to its high temporal and spatial resolution, as well as its low cost, portability, lack of radiation, and ability to evaluate anatomic and functional information [4]. Spatial resolution better than 100 μm and temporal resolution better than 600 frames/sec are now available for practical applications. Moreover, real-time, 3D color Doppler imaging can provide quantifying blood flow in the heart. However, echocardiography has several pitfalls, such as a poor sonic window in postoperative (sternotomy or mechanical valves) or obese patients and suboptimal evaluation of aorta and pericardial diseases [5]. Consequently, many patients undergo additional cardiac CT examination to evaluate cardiac lesions.

Although CT does not demonstrate real-time images, recent advancement of CT equipment and image processing methods have provided significant improvements in temporal resolution. Because of the limitation of temporal resolution on cardiac CT, beta-blocker medication is often used to reduce heart rate during the CT acquisition. Also, considering that the spatial resolution of CT is reached at 0.3–0.4 mm, nitroglycerin is sometimes given to patients to visualize small coronary arteries. The ability of cardiac imaging techniques relates to capture image detail (spatial resolution) during the shortest time expressed as temporal resolution (milliseconds). Fast gantry rotation speed allows capture of a large amount of data, which are necessary to evaluate the regional wall motion of the heart in a single cardiac cycle. Because an image can be reconstructed using 180° of CT acquisition data, the temporal resolution would be 250 ms in a scanner with 500 ms rotation speed.

**CT PROTOCOL**

The postoperative heart CT protocol is similar to that of routine coronary CT angiography, but it can be modified to assess the sternotomy site, valve position and motion, and periaortic/paraaortic complications [5]. We have routinely used retrospective ECG-gated CT to avoid motion artifacts and to achieve adequate image quality for the evaluation of coronary arteries and reconstruction of valve images. Evaluation should include the location and multiphase motion of the valve, potential thrombus (acute stage in the setting of inadequate anticoagulation) and thickening, and calcification and pannus formation around the valve. Coronary arteries are also evaluated not only for potential atheroma, but also for iatrogenic obstruction due to valve implant. Paravalvular structures including the aortic root and left ventricular outflow tract (LVOT) are assessed to determine whether there is any cause of obstruction. Immediate mediastinal complications, such as chest wall or sternal dehiscence, mediastinitis with or without complicated fluid, and source of hemothorax or postoperative bleeding, are also evaluated. In patients with suspected mediastinitis or an abscess, we perform additional 1-min delayed CT scans after obtaining routine coronary CT scans.

**Paravalvular aortic regurgitation**

Paravalvular aortic regurgitation is a potentially serious complication of AVR. It is the regurgitation of blood flow between the prosthetic ring and native annulus due to incomplete sealing. The reported incidence of paravalvular aortic regurgitation is 2–10% after valve replacement [6,7]. Most paravalvular aortic regurgitation cases are trivial or mild asymptomatic regurgitations that are incidentally detected on intraoperative transesophageal echocardiography (TEE) or postoperative transthoracic echocardiography (TTE) [8]. However, approximately 1–5% of patients progress to severe paravalvular aortic regurgitation, which occurs more frequently in the mitral valve than in the aortic valve [9,10].

Abnormal pressure or traction forces on the prosthesis can

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**Table 1. Complications after aortic valve replacement**

<table>
<thead>
<tr>
<th>Complications</th>
<th>Rate (%)</th>
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<tbody>
<tr>
<td>Early</td>
<td></td>
</tr>
<tr>
<td>Paravalvular aortic regurgitation</td>
<td>30-day rate: 0.9</td>
</tr>
<tr>
<td></td>
<td>1-year rate: 0.5</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>0.6</td>
</tr>
<tr>
<td>Coronary artery occlusion (stenosis)</td>
<td>0.3–5</td>
</tr>
<tr>
<td>Stroke</td>
<td>7–17</td>
</tr>
<tr>
<td>Mediastinitis</td>
<td>0.9–20*</td>
</tr>
<tr>
<td>Late</td>
<td></td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>1–6</td>
</tr>
<tr>
<td>Pannus formation</td>
<td>NA</td>
</tr>
<tr>
<td>Sternal osteomyelitis</td>
<td>1–3*</td>
</tr>
<tr>
<td>Paravalvular aortic regurgitation</td>
<td>0.1–1.3</td>
</tr>
<tr>
<td>Hemolysis</td>
<td>5–15</td>
</tr>
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*rates obtained from patients who underwent heart surgery, not only aortic valve surgery. NA: not available
cause incomplete sealing of the prosthetic valve after surgery [11]. The known risk factors for prosthetic valve dehiscence include bacterial endocarditis, aneurysm of the ascending aorta, and diffuse calcification of the native aortic valve. In addition, infection, suturing technique, and size and shape of the prosthesis are known to affect the risk of leaking [12,13]. The most common cause of dehiscence is bacterial endocarditis [14]. Early development of paravalvular aortic regurgitation can be associated with surgical technique and prostheses, and late development of paravalvular aortic regurgitation is mainly caused by dehiscence related to infective endocarditis [13]. With time, the inflammation extends from the prosthetic valve into the paravalvular tissue, leading to tissue destruction and necrosis. Without prompt diagnosis and proper treatment, destruction of the annulus leads to valvular dehiscence and paravalvular aortic regurgitation, which are serious complications [15]. The clinical manifestations of paravalvular aortic regurgitation depend on the severity of regurgitation, ranging from asymptomatic cases to congestive heart failure and hemolytic anemia. Paravalvular aortic regurgitation should be suspected in a patient with a new heart murmur [16].

Echocardiography is regarded as the gold standard modality to evaluate paravalvular aortic regurgitation. Due to acoustic artifacts caused by the prosthetic valve, TEE, rather than TTE, is used to assess the site and severity of paravalvular leakage. Three-dimensional TEE has better diagnostic accuracy than two-dimensional TEE in patients with multiple defects [13]. Although cardiac CT cannot directly visualize regurgitation flow, it can accurately demonstrate the site and morphology of the leaks using multiplanar reconstruction and volume rendering techniques (Fig. 1) and can guide percutaneous repair to avoid repeat valve surgery [17,18]. By three-dimensional reconstruction, we can accurately determine the size, location, and shape of the dehiscence to facilitate surgical planning [19]. On CT images, dehiscence is seen as a gap between the aortic annulus and margin of the prosthetic valve through which contrast material flows from the left ventricular chamber into the aortic root (Fig. 2). It is well visualized on LVOT multiplanar reformatted images [12,20]. Surgery has been the traditional treatment for symptomatic paravalvular aortic regurgitation; however, percutaneous transcatheter closure has been performed with technical success rates of 77–86% [21,22].
Thrombosis and pannus formation

Prosthetic valve obstruction should be suspected in patients with increased transvalvular gradient following AVR or new symptoms of valve obstruction, such as dyspnea or heart failure. The most common causes of prosthetic valve obstruction are thrombosis and pannus formation [23,24].

The reported prevalence of symptomatic obstructive thrombosis in mechanical valves ranges from 0.3% to 1.3%, but subclinical (obstructive or nonobstructive) thrombosis is found in as many as 10% of cases [25]. The most important cause of prosthetic valve thrombosis is inadequate anticoagulation, which is more common with mechanical valves, but thrombosis can occur with bioprostheses long after implantation [23,26,27].

Pannus formation is a chronic complication after AVR, caused by fibrous tissue ingrowth around the prosthetic valve. The reported incidence of pannus formation is 0.2–4.5% [28,29]. In a surgical study, the incidence of pannus formation in combination with thrombus was 12% among 112 obstructed mechanical valves [28].

It is important to distinguish between thrombosis and pannus formation as the cause of valve occlusion. Thromboses can be treated with thrombolysis, whereas pannus formation requires surgical intervention. Although TTE and TEE are useful modalities for evaluating the severity of valve obstruction, they are less suitable for aortic valve obstruction because of acoustic shadowing, particularly if the mass is located on the anterior side of the aortic prosthetic valve [30,31].

Multidetector CT is useful to distinguish thrombus from

Fig. 3. Four years after surgical aortic valve replacement, a 49-year-old woman developed dyspnea. (A) On an aortic valve in-plane view, subvalvular tissue ingrowth (arrows) below the prosthetic aortic valve is noted. (B) A sagittal image of the left ventricular outflow tract shows the lesion in the subvalvular area (arrows). The lesion is showing 160–220 Hounsfield units on computed tomography, suggesting pannus formation. Transvalvular peak velocity (3.4 m/s) and pressure gradient (45/25 mm Hg) were high on echocardiography. AO: ascending aorta, LA: left atrium, LV: left ventricle, RVOT: right ventricular outflow tract.

Fig. 4. A 65-year-old man who underwent surgical aortic valve replacement 9 years previously developed chest discomfort. The INR was 0.94 at the time of admission. (A) On echocardiography, a low-echoic movable lesion (white arrow) is visible with left prosthetic leaflet dysfunction. (B) The lesion (white arrow) shows low attenuation (52 Hounsfield units) on computed tomography and was suspected to be a thrombus. After adjustment for optimal anticoagulation, the INR increased to 2.3, and the lesion was no longer visible on follow-up echocardiography after 3 months (not shown). AO: aorta, LA: left atrium, LV: left ventricle, INR: international normalized ratio.
pannus formation in prosthetic valves. Hounsfield unit of the lesions can be used to distinguish thromboses from pannus, as pannus tend to show higher attenuation. Gunduz et al. [32] reported that thromboses could be distinguished from pannus with high sensitivity (87.5%) and specificity (95.5%) using a cut-off point of 145 Hounsfield units. A pannus is shown as a soft tissue attenuated lesion with a circular anatomical configuration along the subvalvular area to the valve ring (Fig. 3). In contrast, a thrombus is shown as a low attenuated lesion with irregular anatomy directly attached to leaflets and hinge points causing restriction of leaflet motion [31,33-35] (Fig. 4).

**Sternal osteomyelitis and mediastinitis**

Sternal osteomyelitis occurs in 1–3% of patients who undergo sternotomy for heart surgery [36,37]. This condition can cause poststernotomy mediastinitis, which is a severe complication with a mortality rate of approximately 29% [37]. Its risk factors include obesity, diabetes, chronic obstructive pulmonary disease, chronic cough, steroid therapy, and internal mammary artery grafts [38]. Common CT findings in sternal osteomyelitis include bone destruction with demineralization and dehiscence [39]. Because aseptic hematoma or fluid collection in the mediastinum can be seen 2–3 weeks after sternotomy, it is not always easy to differentiate normal findings from those of infectious mediastinitis and abscess formation [40]. If mediastinal fluid collection with air persists for 2 weeks after operation, with redness and discharge from the sternotomy site, infectious mediastinitis should be suspected [41]. On CT images, mediastinal fluid collection with a thick enhancing peripheral rim, soft tissue edema, and sternal closure defects are suggestive of mediastinitis [42] (Fig. 5).

**Infective endocarditis**

The incidence of prosthetic valve endocarditis (PVE) is not especially low: it affects 1–6% of patients [43]. Although new diagnostic and therapeutic strategies have been introduced, high mortality rates of 19–40% from aortic PVE have been reported [15,44,45]. PVE with onset within 2 months after surgery is classified as early PVE, while PVE that develops 12 months or more after surgery is late PVE. PVE occurring 2–12 months after surgery is classified as intermediate PVE [46,47]. Aortic PVE usually originates at the sewing cuff or at thrombi near the sewing ring [48]. Progression of the infection into the perivalvular tissue and abscess formation are more likely to occur with PVE than with native endocarditis [15,43] (Fig. 6). Abscesses, pseudoaneurysm formation, and valve dehiscence are complications of PVE that can occur in approximately 60% of patients [12,47].

TTE and TEE are performed as part of routine surveillance in order to monitor complications in patients with AVR. However, in patients with prosthetic valves, TTE and TEE play a limited role in the diagnosis of PVE compared with native valve endocarditis. TTE has a reported sensitivity of 36% and a specificity of 69%; TEE has a reported sensitivity of 82% and a specificity of 88% [15,47,49].

Cardiac CT has an emerging role as a complementary diagnostic modality for PVE. The sensitivities of CT and TEE to detect vegetation are not significantly different for surgically confirmed infective endocarditis (100% in TEE and 90.9–96% in CT), and the perivalvular extent of abscess/pseudoaneurysms is more clearly demonstrable on CT than on TEE [50,51]. CT can provide a more accurate visualization of the anatomy to evaluate the extent and location of disease [52].

**Embolic events**

Thromboembolism can be caused by a thrombus on a prosthetic valve. Left side valve (mitral and aortic) thrombus can cause systemic embolic events such as stroke and myocardial infarction. The incidence of thromboembolism during the first 3 months after prosthetic valve replacement is reported to be approximately 3.6–6%, which is significantly higher than the incidence after that period [53,54]. The long-term incidence of systemic embolization is approximately 0.7–1.0% in patients...
Fig. 6. Four months after surgical aortic valve replacement, a 58-year-old man with fever and chills visited the emergency department. (A) On transesophageal echocardiography, a small, low-echogenic lesion (white arrow) suggesting an abscess is visible in the thickened echogenic inflamed periaortic area (asterisk). A small movable echogenic focus (black arrow) attached to the prosthetic aortic valve is also seen. (B) Aortic valve in-plane view of computed tomography image also shows a small paravalvular abscess (arrow) and slightly increased soft tissue attenuation (asterisks) in the paravalvular area. (C) Mitral aortic intervalvular fibrosa thickening (asterisk) and internal low echogenicity (white arrow) are visible on the left ventricular outflow tract view. (D) On computed tomography, corresponding areas of inflammation (asterisk) and abscess (white arrow) involving mitral aortic intervalvular fibrosa are evident. A small, low-attenuation (black arrow) vegetation is attached to the prosthetic valve. (E) Surgical specimen revealed vegetation (black arrow) and fibrous thickening of the aortic tissue valve. AO: aorta, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle, RVOT: right ventricular outflow tract.

Fig. 7. A 72-year-old man who underwent a Bentall operation with prosthetic aortic valve replacement 6 days earlier due to ascending aorta aneurysm with aortic regurgitation. (A) The patient presented with chest pain, and postoperative electrocardiography showed abnormal II, III, and aVF elevations suggesting acute inferior wall infarction. (B and C) On postoperative computed tomography, myocardial low-attenuation areas in the mid to basal inferior and inferolateral wall (arrows) are noted, suggesting myocardial ischemic insults.
Coronary artery embolism is a rare cause of myocardial infarction (Fig. 7). The risk factors for coronary artery embolism include prosthetic heart valves with subtherapeutic anticoagulation, infective endocarditis, atrial fibrillation, and atrial myxomas [57]. Most cases of coronary emboli involve the left coronary artery due to preferential flow related to aortic valve morphology [58].

Aortic dissection

Type I aortic dissection after AVR occurs in approximately 0.6% of patients within 1 month to 16 years after surgery, with a poor outcome in 50% of cases [12]. Fragility of the aortic wall, aortic regurgitation, and aortic wall thinning were identified as independent risk factors for dissection after surgery; however, previous aortic cross-clamping or cannulation or type of prosthesis were not associated factors [59]. In particular, AVR of the bicuspid valve was associated with greater incidence of sudden death and delayed acute aortic complications [60].

CT is the diagnostic choice to evaluate aortic dissection with high sensitivity, high specificity, and availability. A negative TTE is not a guaranty of missed dissection, and, considering the high mortality rate of aortic dissection, CT is necessary to evaluate the whole aorta in order to exclude disease. Even a positive TTE can be followed by CT to evaluate the involved extent and assist preoperative planning. In CT images, aortic dissection can be diagnosed by identifying an intimal flap separating the false and true lumens [61], with a reported diagnostic accuracy of 88–100% [62] (Fig. 8). Ulcer-like projections, displaced intimal calcifications, and compression of the true lumen are additional aortic dissection findings. A lumen with a larger cross section and beak sign usually indicates a dissection-related false lumen [62].

CONCLUSION

Multidetector CT imaging is becoming popular as a diagnostic modality complementary to echocardiography. CT allows objective anatomic visualization of valvular as well as perivalvular lesions, which is useful for treatment planning, accurate diagnosis, and follow-up.

Conflicts of Interest

The authors declare that they have no conflict of interest.

REFERENCES

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